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A. B. McCormack A. L. Li J. Uversky V. N. Fink A. L. Di Monte D. A. Manning-Bog. The herbicide paraquat causes up-regulation and aggregation of alpha-synuclein in mice: paraquat and alpha-synuclein. <i>J Biol Chem.</i> 2001. 277:1641-4
J. Stevenson F. F. Oo M. L. Andersen J. K. Peng. Iron-enhanced paraquat-mediated dopaminergic cell death due to increased oxidative stress as a consequence of microglial activation. <i>Free Radic Biol Med.</i> 2008. 46:312-20
S. E. Gremaud J. N. Skagen K. Steed E. Dalton R. Sugden K. D. Cardozo-Pelaez F. Sherwin C. M. Woodahl E. L. Lacher. Absence of P-glycoprotein transport in the pharmacokinetics and toxicity of the herbicide paraquat. <i>J Pharmacol Exp Ther.</i> 2013. 348:336-45
R. Godena V. K. Sanchez-Martinez A. Ferrari E. Casella L. Beltramini M. Bubacco L. Whitworth A. J. Bisaglia M. Filograna. Superoxide Dismutase (SOD)-mimetic M40403 Is Protective in Cell and Fly Models of Paraquat Toxicity: IMPLICATIONS FOR PARKINSON DISEASE. <i>J Biol Chem.</i> 2016. 291:9257-67
J. Anandhan Navarro-Yepes. Inhibition of Protein Ubiquitination by Paraquat and 1-Methyl-4-Phenylpyridinium Impairs Ubiquitin-Dependent Protein Degradation Pathways. <i>Mol Neurobiol.</i> 2015. #volume#:#pages#
J. Mao Peng. The herbicide paraquat induces dopaminergic nigral apoptosis through sustained activation of the JNK pathway. <i>J Biol Chem.</i> 2004. 279:32626-32
H. S. An Choi. PEP-1-SOD fusion protein efficiently protects against paraquat-induced dopaminergic neuron damage in a Parkinson disease mouse model. <i>Free Radic Biol Med.</i> 2006. 41:1058-68
A. C. Choi Cristovao. The role of NADPH oxidase 1-derived reactive oxygen species in paraquat-mediated dopaminergic cell death. <i>Antioxid Redox Signal.</i> 2009. 11:2105-18
E. N. Peters Mangano. Granulocyte macrophage-colony stimulating factor protects against substantia nigra dopaminergic cell loss in an environmental toxin model of Parkinson's disease. <i>Neurobiol Dis.</i> 2011. 43:99-112
P. M. Cui Rappold. Paraquat neurotoxicity is mediated by the dopamine transporter and organic cation transporter-3. <i>Proc Natl Acad Sci U S A.</i> 2011. 108:20766-71
A. C. Guhathakurta Cristovao. NADPH oxidase 1 mediates alpha-synucleinopathy in Parkinson's disease. <i>J Neurosci.</i> 2012. 32:14465-77
F. Zhao, W. Wang, C. Wang, S. L. Siedlak, H. Fujioka, B. Tang, X. Zhu. Mfn2 Protects Dopaminergic Neurons Exposed to Paraquat Both in vitro and in vivo: Implications for Idiopathic Parkinson's Disease. <i>Biochim Biophys Acta.</i> 2017. #volume#:#pages#
J. Peng L. Stevenson F. F. Doctrow S. R. Andersen J. K. Peng. Iron and paraquat as synergistic environmental risk factors in sporadic Parkinson's disease accelerate age-related neurodegeneration. <i>J Neurosci.</i> 2007. 27:6914-22
J. Oo M. L. Andersen J. K. Peng. Synergistic effects of environmental risk factors and gene mutations in Parkinson's disease accelerate age-related neurodegeneration. <i>J Neurochem.</i> 2010. 115:1363-73
E. Parafati Janda. The antidote effect of quinone oxidoreductase 2 inhibitor against paraquat-induced toxicity in vitro and in vivo. <i>Br J Pharmacol.</i> 2013. 168:46-59
H. Garcia Garcia Rodriguez-Rocha. Glutaredoxin 1 protects dopaminergic cells by increased protein glutathionylation in experimental Parkinson's disease. <i>Antioxid Redox Signal.</i> 2012. 17:1676-93
F. Lapointe Cicchetti. Systemic exposure to paraquat and maneb models early Parkinson's disease in young adult rats. <i>Neurobiol Dis.</i> 2005. 20:360-71

alpha-Synuclein-containing aggregates represent a feature of a variety of neurodegenerative disorders, including Parkinson's disease (PD).
Environmental paraquat and neonatal iron exposure have both separately been suggested as potential risk factors for sporadic PD.
Genetic variation in the multidrug resistance gene ABCB1, which encodes the efflux transporter P-glycoprotein (P-gp), has been suggested as a potential risk factor for PD.
Parkinson disease is a debilitating and incurable neurodegenerative disorder affecting approximately 1-2% of people over 65 years of age.
Intracytoplasmic inclusions of protein aggregates in dopaminergic cells (Lewy bodies) are the pathological hallmark of Parkinson's disease (PD).
Environmental exposure to the oxidant-producing herbicide paraquat has been implicated as a risk factor in Parkinson's disease (PD).
Parkinson disease (PD) is a common neurodegenerative disorder characterized by the progressive loss of dopaminergic neurons in the substantia nigra.
Oxidative stress is the common downstream effect of a variety of environmental neurotoxins that are strongly implicated in the pathogenesis of PD.
Parkinson's disease (PD) has been linked to exposure to a variety of chemical (e.g., pesticides) and inflammatory agents, suggesting a multifactorial etiology.
The herbicide paraquat (PQ) has increasingly been reported in epidemiological studies to enhance the risk of developing PD.
Accumulation of misfolded alpha-synuclein is the pathological hallmark of Parkinson's disease (PD). Nevertheless, little is known about the mechanisms of alpha-synuclein misfolding and aggregation.
Mitochondrial dynamics and quality control plays a critical role in the maintenance of mitochondrial homeostasis and function, and is implicated in the pathogenesis of PD.
Extensive epidemiological data in humans and studies in animal models of Parkinson's disease (PD) suggest that sporadic PD is a multifactorial disorder.
As Parkinson's disease appears to be a multifactorial disorder, the use of animal models to investigate combined effects of environmental and genetic factors is essential.
BACKGROUND AND PURPOSE The mechanisms of paraquat (PQ)-induced toxicity are poorly understood and PQ poisoning remains a significant public health problem.
AIMS: Chronic exposure to environmental toxicants, such as paraquat, has been suggested as a risk factor for Parkinson's disease (PD).
In recent years, several lines of evidence have shown an increase in Parkinson's disease (PD) prevalence in rural environments, particularly in areas with high levels of paraquat use.

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W. S. Abel Choi. JNK3 mediates paraquat- and rotenone-induced dopaminergic neuron death. <i>J Neuropathol Exp Neurol.</i> 2010. 69:511-20
R. Ganfornina Bajo-Graneras. Apolipoprotein D mediates autocrine protection of astrocytes and controls their reactivity level, contributing to the functional maintenance of paraquat-challenged dopaminergic systems. <i>Glia.</i> 2011. 59:1551-66
H. Wu Li. Neuroprotective effects of tert-butylhydroquinone on paraquat-induced dopaminergic cell degeneration in C57BL/6 mice and in PC12 cells. <i>Arch Toxicol.</i> 2012. 86:1729-40
M. Hichor, N. K. Sampathkumar, J. Montanaro, D. Borderie, P. X. Petit, V. Gorgievski, E. T. Tzavara, A. A. Eid, F. Charbonnier, J. Grenier, C. Massaad. Paraquat Induces Peripheral Myelin Disruption and Locomotor Defects: Crosstalk with LXR and Wnt Pathways. <i>Antioxid Redox Signal.</i> 2016. #volume#: #pages#
R. Ganfornina L. Sánchez D. Bajo. Apolipoprotein D mediates autocrine protection of astrocytes, contributing to the functional maintenance of paraquat-challenged dopaminergic systems. <i>GLIA.</i> 2011. 59:S70
E. Casali C. Caporali M. Sancesario G. Morocutti C. Stefano. Parkinson disease in farm workers. <i>Ital J Neurol Sci.</i> 1989. 10:379
P. J. Van Vliet C. Borm. Susceptibility in Parkinson. <i>Med Hypotheses.</i> 1988. 27:205-7
J. R. Hefti F. Weiner W. J. Sanchez-Ramos. Paraquat and Parkinson. <i>Neurology.</i> 1987. 37:728
A. H. Uitti R. J. Rajput. Paraquat and Parkinson. <i>Neurology.</i> 1987. 37:1820-1
A. H. Uitti R. J. Stern W. Laverty W. O. Geography, drinking water chemistry, pesticides and herbicides and the etiology of Parkinson. <i>Can J Neurol Sci.</i> 1987. 14:414-8
W. C. Koller. Paraquat and Parkinson. <i>Neurology.</i> 1986. 36:1147
T. Tawara T. Isobe A. Hojo N. Shiwaku K. Yamane Y. Fukushima. Radical formation site of cerebral complex I and Parkinson. <i>J Neurosci Res.</i> 1995. 42:385-90
M. C. McLean A. J. Rivory L. P. Le Couteur D. G. Yang. Hepatic disposition of neurotoxins and pesticides. <i>Pharmacol Toxicol.</i> 2001. 87:286-91
M. C. McLean A. J. Le Couteur D. G. Yang. Age-related alteration in hepatic disposition of the neurotoxin 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine and pesticides. <i>Pharmacol Toxicol.</i> 2002. 90:203-7
J. K. Andersen. Paraquat and iron exposure as possible synergistic environmental risk factors in Parkinson. <i>Neurotox Res.</i> 2004. 5:307-13
S. Sinha A. Singh M. P. Patel. Identification of differentially expressed proteins in striatum of maneb- and paraquat-induced Parkinson's disease phenotype in mouse. <i>Neurotoxicol Teratol.</i> 2007. 29:578-85
S. Singh K. Singh S. Singh M. P. Patel. Gene expression profiles of mouse striatum in control and maneb + paraquat-induced Parkinson's disease phenotype: validation of differentially expressed energy metabolizing transcripts. <i>Mol Biotechnol.</i> 2008. 40:59-68
B. R. Manthripragada A. D. Costello S. Lincoln S. J. Farrer M. J. Cockburn M. Bronstein J. Ritz. Dopamine transporter genetic variants and pesticides in Parkinson's disease. <i>Environ Health Perspect.</i> 2009. 117:964-9
A. Irizarry M. C. Schwarzschild M. A. Kachroo. Caffeine protects against combined paraquat and maneb-induced dopaminergic neuron degeneration. <i>Exp Neurol.</i> 2010. 223:657-61
R. A. Niso-Santano M. Gomez-Sanchez R. Bravo-San Pedro J. M. Fuentes J. M. Gonzalez-Polo. DJ-1 as a modulator of autophagy: an hypothesis. <i>ScientificWorldJournal.</i> 2010. 10:1574-9
R. M. Murali D. Nickles R. J. Barnhart T. E. Holden J. E. DeJesus O. T. Bartlett. Assessment of fetal brain uptake of paraquat in utero using in vivo PET/CT imaging. <i>Toxicol Sci.</i> 2011. 122:551-6

Mechanistic studies underlying dopaminergic neuron death may identify new drug targets for the treatment of Parkinson's disease.
The study of glial derived factors induced by injury and degeneration is important to understand the nervous system response to injury.
The present study was aimed at determining the role of paraquat (PQ) in the activation of the NF-E2-related factor 2 (Nrf2) pathway.
AIMS: Paraquat (PQT), a redox-active herbicide, is a free radical-producing molecule, causing damage particularly to the nervous system.
Oxidative stress, concomitant to injury and degeneration in the CNS, triggers the release of many glial derived factors. Apoptosis is a key event in the pathogenesis of Parkinson's disease.
Nowadays, a substantial amount of clinical and experimental research is directed to the role of reactive oxygen species in the pathogenesis of Parkinson's disease.
In 1984 we made the first observation of a correlation between early age exposure to rural environment (and drinking water) and the risk of developing Parkinson's disease.
Paraquat was reduced to the paraquat radical via complex I in bovine cerebral mitochondria and accelerated lipid peroxidation.
The hepatic disposition of pesticides and neurotoxins may influence susceptibility to Parkinson's disease. Therefore we evaluated the effect of paraquat on the hepatic disposition of pesticides.
Idiopathic Parkinson's disease may be caused by environmental neurotoxins such as pesticides, however the major risk factor remains unknown.
Behavioral, phenotypic and biochemical changes induced by maneb+paraquat (MB+PQ) in experimental animals have shown similarities with those observed in Parkinson's disease.
The present study was undertaken to investigate the gene expression patterns of the striatum of control and maneb + paraquat treated mice.
BACKGROUND: Research suggests that independent and joint effects of genetic variability in the dopamine transporter (DAT) and the alpha-synuclein gene (SNCA) may influence the risk of developing Parkinson's disease.
Environmental exposures suspected of contributing to the pathophysiology of Parkinson's disease (PD) include potential neurotoxins such as pesticides, heavy metals, and herbicides.
The etiology of Parkinson's disease (PD) is not completely defined, although environmental factors (for example, exposure to pesticides) are thought to play a role in the development of adult diseases including Parkinson's disease.
Prenatal in utero conditions are thought to play a role in the development of adult diseases including Parkinson's disease.

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A. Spivey. Rotenone and paraquat linked to Parkinson's disease: human exposure study supports years of animal studies. <i>Environ Health Perspect.</i> 2011. 119:A259
K. Kim S. H. Kim J. Kim H. Yim J. Kim. Glutathione s-transferase omega 1 activity is sufficient to suppress neurodegeneration in a Drosophila model of Parkinson disease. <i>J Biol Chem.</i> 2012. 287:6628-41
P. Patel P. Kosberg K. Mante M. Patrick C. Rockenstein E. Fujita M. Hashimoto M. Masliah E. Desplats. Combined exposure to Maneb and Paraquat alters transcriptional regulation of neurogenesis-related genes in mice models of Parkinson's disease. <i>Mol Neurodegener.</i> 2012. 7:49
J. A. Campbell C. Tomenson. How did a reduced incidence of Parkinson's disease become an increase?. <i>Neurotoxicology.</i> 2013. 36:104
J. Brent. High-dose paraquat exposure and Parkinson's disease. <i>Neurotoxicology.</i> 2013. 37:247
F. Kamel. Epidemiology. Paths from pesticides to Parkinson's. <i>Science.</i> 2013. 341:722-3
N. K. Chauhan A. K. Jain S. K. Shanker R. Singh C. Singh M. P. Singhal. Silymarin- and melatonin-mediated changes in the expression of selected genes in pesticides-induced Parkinsonism. <i>Mol Cell Biochem.</i> 2013. 384:47-58
G. Lewis M. M. Sterling N. W. Kong L. Chen H. Mailman R. B. Huang X. Du. Microstructural changes in the substantia nigra of asymptomatic agricultural workers. <i>Neurotoxicol Teratol.</i> 2013. 41:60-4
S. Di Benedetto M. D'Addario C. Candeletti S. Romualdi P. Bastias-Candia. Combined exposure to agriculture pesticides, paraquat and maneb, induces alterations in the N/OFQ-NOPr and PDYN/KOPr systems in rats: Relevance to sporadic Parkinson's disease. <i>Environ Toxicol.</i> 2014. 30:656-63
M. G. Pinheiro A. S. de Souza T. L. Follmer C. Maturana. Unveiling the role of the pesticides paraquat and rotenone on alpha-synuclein fibrillation in vitro. <i>Neurotoxicology.</i> 2014. 46:35-43
E. Leverage W. T. Liu Y. Panzella L. Alfieri M. L. Napolitano A. Bentley W. E. Payne G. F. Kim. Paraquat-Melanin Redox-Cycling: Evidence from Electrochemical Reverse Engineering. <i>ACS Chem Neurosci.</i> 2016. 7:1057-67
Anon. Paraquat and Parkinson's disease. <i>Food and Chemical Toxicology.</i> 1997. 35:929
D. Xu He. 3.108 ELECTROPHYSIOLOGY OF NEOSTRIATAL NEURONS IN A RAT MODEL OF PARKINSON'S DISEASE INDUCED BY PARAQUAT AND MANEB. <i>Parkinsonism & Related Disorders.</i> 2012. 18, Supplement 2:S191
Shin-ichi Kyuhou. Preventive effects of phytoestrogens on the paraquat-induced toxicities in the cellular model of Parkinson disease. <i>Neuroscience Research.</i> 2007. 58, Supplement <HT>1</HT>:S234
Enda Cindylosa Adji Sitepu. Neuroprotective effect of ethanolic extract of Pandanus Odoratissimus Ln. in Paraquat mice model of Parkinson's disease. <i>Parkinsonism & Related Disorders.</i> 2016. 22, Supplement 2:e187
K. Sandhu J. K. Sikorska M. Somayajulu-Nitu M. Dadwal P. Rafo N. Church K. Cohen J. Borowy-Borowski H. Pandey S. Weinstock S. Facecchia. Protection of SNpc neurons by water soluble CoQ(10) in a paraquat induced rat model of Parkinson's disease: The role of neurotrophic factors. <i>Faseb Journal.</i> 2010. 24:#pages#
A. Ceravolo R. Dell'Agnello G. Gambaccini G. Bellini G. Kiferle L. Rossi C. Logi C. Bonuccelli U. Nuti. Environmental factors and Parkinson's disease: a case-control study in the Tuscany region of Italy. <i>Parkinsonism & Related Disorders.</i> 2004. 10:481-485
J. M. Keller-Byrne J. Wright. Environmental determinants of Parkinson's disease. <i>Archives of Environmental Health.</i> 2005. 60:32-38

A loss-of-function mutation in the gene parkin causes a common neurodegenerative disease that may be caused by mito
BACKGROUND: Parkinson's disease (PD) is a multifactorial disease where environmental factors act on genetically predis
Parkinson's disease (PD) is the second most unconcealed neurodegenerative disorder labelled with motor impairments.
Parkinson's disease (PD) is marked by the loss of dopamine neurons in the substantia nigra (SN). Although the exact etiol
Despite several years of research, the aetiology of Parkinson's disease (PD) is quite far from being solved. In PD, as well a
Epidemiological data have suggested that exposure to environmental toxins might be associated with the etiology of Par
Parkinson's disease is a neurodegenerative disorder associated with oxidative stress and the death of melanin-containing
To date the aetiology of Parkinson's disease (PD) is unknown although both genetic susceptibility and environmental fact
Increasing toxicologic and epidemiologic evidence suggests that pesticides and other environmental exposures are assoc

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